Periscope.

CLINICAL NEUROLOGY.

142 UEBER DIE BEZIEHUNGEN EPILEPTISCHER ANFÄLLE ZUR HARNSAURE-AUSSCHEIDUNG (On the Relations Subsisting Between Epileptic Attacks and the Excretion of Uric Acid). Caro (Deutsche med. Woch., May 10, 1900).

Caro first recapitulates the work of his predecessors in this direction. In 1896 Haig noted that before epileptic attacks there was a diminution in the excretion of uric acid. In 1897 Kieman found that, generally speaking, the amount of uric acid in the blood of epileptics was increased, while Charon and Briche found that in certain cases there was a connection between the alkalinity of the blood and severe epileptic attacks; with fewer and milder attacks there was increased alkalescence of blood, and vice versa. Recently Krainsky has made some important studies; he found 24 to 48 hours before an attack, a regular diminution of acid in the urine, so regular, in fact, as to have a prognostic value. Krainsky regards the diminished amount af acid as due to a toxic action. This poisonous substance causes the convulsion; and is in turn destroyed by it. The blood of epileptics is toxic to animals at or just before the attack. These findings confirm the results given by others. Krainsky's supposition was that the toxic agent is ammonium *carbamate,* with free ammonia in the blood.

Caro gives a curve which shows the percentage of uric acid and its relation to several epileptic attacks occurring within a month. Epilepsy must be conceived at the present time as a result of intoxication; all other convulsive maladies are of this nature, uremia, eclampsia, convulsions in ordinary poisoning, the convulsions of children attacked by acute infectious diseases. Even the presence of focal disease in the brain does not wholly invalidate this conclusion; for a symptom complex may be caused by different affections. CLARK.

143 EPILEPSY. W. P. Spratling (Buffalo Med. Jour., June, 1900).

In this paper Spratling says that the disease is found most frequently (1) during early infancy and dentition; (2) during the period covered by the seventh and eighth years, and (3) during the marked transitional period of puberty. Among the causes are: (1) direct transmission from parent to child; (2) accidents at birth; (3) intense pathological processes that should be physiological, such as difficult dentition, especially in rachitic or scrofulous children; (4) indigestion and malassimilation; (5) accidents of early life, and (6) the grave physiological disturbances that come at puberty. For prognostic purposes it is essential to divide epilepsy into that form due to some physical defect or deformity and into the true form of epilepsy in which no direct cause is apparent and in which the physician can do far less good. Treatment may be in institutions or at home; in the latter case, too much parental sympathy, the too liberal use of medicines, especially of the proprietary kind, and improper food, often defeat the object in view. It is important that patients should have meat but once a day, and that at noon and only in small quantities; they should have nothing fried in grease and no pies or other pastry. They must eat largely of cereals, milk, fruits, eggs and butter. Of drugs, the fluid extract of horse-nettle berries has given satisfaction, since it does not impair the functions of the digestive tract. Simulo, also, has given excellent results. A combination of bromide, chloral and morphine is often of service in aborting an expected seizure preceded by a long aura. Great stress is to be laid upon the systematic exercise of all the muscles of the patient's body, such work as can best be done being prescribed after a thorough physical examination. JELLIFFE.

144 THE ETIOLOGY AND PATHOLOGY OF MAJOR EPILEPSY. William House (Phila. Med. Jour., Vol. 5, 1900, p. 691).

In this paper the writer reviews briefly the various theories advanced by different observers as to the causation and pathology of epilepsy, and, believing that "like causes produce like effects," compares the symptoms of epilepsy with those of other diseases which produce or are accompanied by convulsive seizures resembling more or less the seizures of epilepsy. These diseases are hysteria, tetany, infantile, puerperal and uremic eclampsia, alcoholism, cerebral hemorrhage, and the apoplectiform and epileptiform convulsions of general paresis. The pathology of the three latter conditions is well known, and in certain respects the lesions are of a similar nature. The symptoms of an epileptic seizure, of an epileptiform paretic convulsion, of an alcoholic convulsion, and of cerebral hemorrhage present a marked similarity, so much so that sometimes it is difficult to make a differential diagnosis. Symptoms of cerebral pressure are present in these three conditions and also in epilepsy. In the brain of an alcoholic there is an excessive quantity of cerebrospinal fluid, the ventricles are distended, the brain substance drips with fluid, and the membranes are dropsical. This is called the "wet brain." In general paresis the ventricles are distended with fluid, there is an increased quantity of fluid in the subdural space, and the whole brain is surrounded with an excessive quantity of turbid cerebrospinal fluid. In both these conditions the excessive fluid seems to the writer to be the logical cause of the pressure symptoms recognized in convulsions from these diseases.

The writer has witnessed the autopsies of five cases of *status epilepticus,* and in each instance there was found an excessive quantity of cerebrospinal fluid.

Arguing from analogy, House is forced to the conviction that this increase in the quantity of cerebrospinal fluid must bear a causal relation to the convulsions of epilepsy. According to physiologic findings, if the cerebrospinal fluid be suddenly withdrawn convulsions may ensue; if rapidly increased coma may be produced.

From the observation of over 200 epileptics, and from the comparison of their symptoms with those of 60 alcoholics and those of a large number of paretics, from the findings in the above five autopsies, and from an analysis of recent literature, he concludes that:

(1) There is no record of pathologic findings which logically explain the symptoms of epilepsy.

(2) An increase of cerebrospinal fluid would readily account for the seizures. In many instances it is analogous to the marked increase of fluid in the crania of alcoholics and paretics, antd is not dissimilar in clinical effects to the more localized lesions of hemorrhage or abscess.

(3) This fluid, physiologically subject to more or less variation in quantity from day to day, is fully capable of pathologic increase, and from analogy must bear exciting relation to the convulsion.

(4) Its increase is probably gradual, and to this we may ascribe the auræ.